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2 NEAGTIVE
0 CD34 NEAGTIVE
(CD34(W)NEAGTIVE)
12294 CD34-
(CD34)
L1 12294 (CD34 NEAGTIVE) OR (CD34-)

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12294 CD34
93072 NEGATIVE
3502 NEGATIVES
95995 NEGATIVE
(NEGATIVE OR NEGATIVES)
612875 NEG
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613055 NEG
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656546 NEGATIVE
(NEGATIVE OR NEG)
114 CD34 NEGATIVE
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12294 CD34-_
(CD34)
L2 12294 (CD34 NEGATIVE) OR (CD34-_)

=> s L2 and (placenta or placental)
44257 PLACENTA
4293 PLACENTAS
1033 PLACENTAE
44995 PLACENTA
(PLACENTA OR PLACENTAS OR PLACENTAE)
32286 PLACENTAL
67 PLACENTALS
32319 PLACENTAL
(PLACENTAL OR PLACENTALS)
L3 209 L2 AND (PLACENTA OR PLACENTAL)

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50217 ANGIOGENESIS

2 VASOGENESIS
L4 23 L3 AND (ANGIOGENESIS OR VASOGENESIS)

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L5 ANSWER 1 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2008:1280223 CAPLUS
DN 149:455416
TI Treatment of insulin resistance and diabetes by increasing skeletal muscle perfusion through administration of cells stimulatory of angiogenesis or vascular responsiveness
IN Riordan, Neil; Ichim, Tom
PA Medistem Labortories, USA
SO U.S. Pat. Appl. Publ., 17pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20080260703	A1	20081023	US 2008-108114	20080423

PRAI US 2007-913533P P 20070423
AB Disclosed are methods, compns., and cells useful for increasing insulin sensitivity, as well as lack of insulin prodn. in a host in need thereof. One aspect of the invention discloses methods of increasing skeletal muscle perfusion through administration of cells capable of directly and/or indirectly stimulatory of angiogenesis and/or vascular responsiveness. Another aspect provides means of increasing sensitivity to insulin through administration of a cell compn. capable of integrating into host insulin responsive tissue and upregulating responsiveness either through mobilization of host cells capable of responding to insulin, mobilization of host cells capable of endowing insulin responsiveness on other host cells, exogenously administered cells taking the role of insulin responsiveness, or exogenously administered cells endowing insulin responsiveness on other host cells. Another aspect comprises modifying said host to allow for concurrent insulin sensitization and upregulated prodn. of insulin. Thus, group of 100 patients were recruited with type 2 diabetes receiving daily insulin injections; 50 patients were treated with placebo control and 50 receive allogeneic menstrual blood derived endometrial regenerative cells (ERC). Patients in the treated group

displayed an increased responsiveness to insulin starting 2 wk after injection of cells.

L5 ANSWER 2 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2008:1251768 CAPLUS
DN 149:432561
TI Stem cell therapy for the treatment of autism and other disorders
IN Riordan, Neil H.; Ichim, Thomas E.
PA Medistem Labortories, USA
SO U.S. Pat. Appl. Publ., 7pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 20080254005	A1	20081016	US 2008-98420	20080405
PRAI US 2007-910605P	P	20070406		

AB Disclosed are methods, compns. of matter, and cells, useful for the treatment of autism, social integrative disorders, and various cognitive abnormalities. The invention discloses, inter alia, means of inducing angiogenesis and immune modulation either in sequence or parallel in order to substantially ameliorate or reverse the progression of autism. The use of stem cells, and cells naturally possessing or endowed with angiogenic and anti-inflammatory activity are disclosed for autism either alone or in combination with various therapeutic interventions.

L5 ANSWER 3 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2008:510664 CAPLUS
TI Characterization of an acyl-coenzyme A binding protein predominantly expressed in human primitive progenitor cells
AU Soupene, Eric; Serikov, Vladimir; Kuypers, Frans A.
CS Children's Hospital Oakland Research Institute, Oakland, CA, 94609, USA
SO Journal of Lipid Research (2008), 49(5), 1103-1112
CODEN: JLPRAW; ISSN: 0022-2275
PB American Society for Biochemistry and Molecular Biology, Inc.
DT Journal
LA English
AB Human acyl-CoA binding domain-contg. member 6 (ACBD6) is a modular protein that carries an acyl-CoA binding domain at its N terminus and two ankyrin motifs at its C terminus. ACBD6 binds long-chain acyl-CoAs with a strong preference for unsatd., C18:1-CoA and C20:4-CoA, over satd., C16:0-CoA, acyl species. Deletion of the C terminus, which is not conserved among the members of this family, did not affect the binding capacity or the substrate specificity of the protein. ACBD6 is not a ubiquitous protein, and its expression is restricted to tissues and progenitor cells with

functions in blood and vessel development. ACBD6 was detected in bone marrow, spleen, placenta, cord blood, circulating CD34 + progenitors, and embryonic-like stem cells derived from placenta . In placenta, the protein was only detected in CD34+ progenitor cells present in blood and in CD31+ endothelial cells surrounding the blood vessels. These cells were also pos. for the marker CD133, and they probably constitute hemangiogenic stem cells, precursors of both blood and vessels. We propose that human ACBD6 represents a cellular marker for primitive progenitor cells with functions in hematopoiesis and vascular endothelium development.

RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2007:1421968 CAPLUS

DN 148:52286

TI Methods and compositions for the treatment of neuropathy using cells which can differentiate into endothelial cells

IN Yoon, Young-Sup

PA Caritas St. Elizabeth Medical Center of Boston, Inc., USA

SO PCT Int. Appl., 87pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2007142651	A1	20071213	WO 2006-US22624	20060609

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI WO 2006-US22624 20060609

AB The invention features compns. and methods that are useful for the prevention or treatment of neuropathy or for enhancing angiogenesis in a neural tissue. The methodol. of the invention involves administration of cells having the potential to differentiate

into endothelial cells.
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2007:1364220 CAPLUS
DN 148:24846
TI Treatment of disc degenerative disease using cells able to increase angiogenesis alone or in combination with growth factors or a matrix and compositions for same
IN Ichim, Thomas E.
PA Medistem Laboratories, Inc., USA
SO PCT Int. Appl., 76pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2007136673	A2	20071129	WO 2007-US11778	20070518
WO 2007136673	A3	20080320		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRAI US 2006-801957P P 20060519
AB Methods and compns. for treating or ameliorating lower back pain by administering an effective amt. of one or more cell types, alone, and/or in combination with a matrix, and/or in combination with growth factors, in order to stimulate lumbar angiogenesis, decrease inflammation, and stimulate regeneration. The cells are substantially composed of stem cells selected from the group consisting of side population, embryonic, germinal, endothelial, hematopoietic, myoblast, placental, cord-blood, adipocyte and mesenchymal stem cells. The cells can be transfected with a gene capable of stimulating angiogenesis, said gene expresses a sol. growth factor. The cells can be programmed to undergo enhanced migration through gene transfection

with receptors for chemotactic ligands. The cells can be used in combination with small interfering RNA specific to an inflammatory stimulus or stimuli to treat lower back pain. The matrix that is administered with the cells is capable of sustaining cellular viability and temporary localization to an injection site.

L5 ANSWER 6 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2007:1064219 CAPLUS
DN 147:383999
TI Detection of gene expression by specific cell types in mixed samples or tissues such as mouse thymus cortex or medullary stromal cells using DGEM (differential gene expression mapping)
IN Petrie, Howard T.
PA USA
SO PCT Int. Appl., 257pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007106507	A2	20070920	WO 2007-US6363	20070314

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI US 2006-782124P P 20060314
AB Differential gene expression mapping (DGEM) utilizes (1) laser capture microdissection or other methods of microdissection of the tissue regions of interest; (2) microarray screening of RNA isolated from the microdissected regions and anal. of purified individual cellular components from the tissue; and (3) computational profiling or subtraction to identify gene expression by specific cell types in situ. The method was applied to stromal cells from whole cortical and medullary regions of C57BL6 mouse thymus. As a result, DGEM, a reverse identification approach, solves previously insurmountable problems, as the lymphoid progenitors can be readily isolated, allowing fluctuations in receptor expression on lymphoid cells to be used to predict stratified stromal

signals. An algorithmic approach can be used for calcg. the expression profile of a tissue/sample of interest that consists of at least two types of cells. Specifically, the approach electronically subtracts the expression profile of one component of a sample from the expression profile of the total sample, thus revealing the profiles of the other component. To confirm the robustness of the DGEM procedure, the gene expression profiles from each sample of whole medulla, whole cortex, cortical thymocytes and medullary thymocytes was sorted based only on the expression data.

L5 ANSWER 7 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2007:410294 CAPLUS
DN 146:377692
TI Proliferation-associated genes/proteins and methods for diagnosis and treatment of proliferation disorders and for drug screening
IN Griffioen, Arjan Willem; Van Beijnum, Judith Rosina
PA Universiteit Maastricht, Neth.
SO PCT Int. Appl., 136pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2007039255	A1	20070412	WO 2006-EP9496	20060929
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2006299079	A1	20070412	AU 2006-299079	20060929
CA 2622852	A1	20070412	CA 2006-2622852	20060929
EP 1928909	A1	20080611	EP 2006-818246	20060929
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
US 20090035312	A1	20090205	US 2008-88670	20080328
PRAI EP 2005-447220	A	20050930		
WO 2006-EP9496	W	20060929		

AB Crucial to designing anti-angiogenic and vascular targeting approaches is

the identification of specific target mols. We compared transcriptional profiles of tumor endothelial cells with that of normal resting endothelial cells, normal but angiogenically activated placental endothelial cells, and cultured endothelial cells. Although the majority of transcripts were classified as general angiogenesis markers, we identified 17 genes that show specific overexpression in tumor endothelium. Antibody targeting of four cell-surface expressed or secreted products (vimentin, CD59, HMGB1 and IGFBP7) inhibited angiogenesis in vitro and in vivo. Finally, targeting endothelial vimentin in a mouse tumor model significantly inhibited tumor growth and reduced microvessel d. Our results demonstrate the utility of the identification and subsequent targeting of specific tumor endothelial markers for anticancer therapy.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2007:638973 CAPLUS

DN 148:75701

TI High-grade clear cell renal cell carcinoma has a higher angiogenic activity than low-grade renal cell carcinoma based on histomorphological quantification and qRT-PCR mRNA expression profile

AU Baldewijns, M. M.; Thijssen, V. L.; Van den Eynden, G. G.; Van Laere, S. J.; Bluekens, A. M.; Roskams, T.; van Poppel, H.; De Bruine, A. P.; Griffioen, A. W.; Vermeulen, P. B.

CS Angiogenesis Laboratory, Department of Pathology, Research Institute for Growth and Development (GROW), & University Hospital Maastricht, Maastricht University, Maastricht, NL 6229 HX, Neth.

SO British Journal of Cancer (2007), 96(12), 1888-1895
CODEN: BJCAAI; ISSN: 0007-0920

PB Nature Publishing Group

DT Journal

LA English

AB Clear cell renal cell carcinoma (CC-RCC) is a highly vascularized tumor and is therefore an attractive disease to study angiogenesis and to test novel angiogenesis inhibitors in early clin. development. Endothelial cell proliferation plays a pivotal role in the process of angiogenesis. The aim of this study was to compare angiogenesis parameters in low nuclear grade (n = 87) vs high nuclear grade CC-RCC (n = 63). A panel of antibodies was used for immunohistochem.: CD34/Ki-67, carbonic anhydrase IX, hypoxia-inducible factor-1.alpha. (HIF-1.alpha.) and vascular endothelial growth factor (VEGF). Vessel d. (MVD - microvessel d.), endothelial cell proliferation fraction (ECP%) and tumor cell proliferation fraction (TCP%) were assessed. mRNA expression levels of angiogenesis

stimulators and inhibitors were detd. by quant. RT-PCR. High-grade CC-RCC showed a higher ECP% (P = 0.049), a higher TCP% (P = 0.009), a higher VEGF protein expression (P < 0.001), a lower MVD (P < 0.001) and a lower HIF-1.alpha. protein expression (P = 0.002) than low-grade CC-RCC. Growth factor mRNA expression analyses revealed a higher expression of angiopoietin 2 in low-grade CC-RCC. Microvessel d. and ECP% were inversely correlated (Rho = -0.26, P = 0.001). Because of the imperfect assocn. of nuclear grade and ECP% or MVD, CC-RCC was also grouped based on low/high MVD and ECP%. This anal. revealed a higher expression of vessel maturation and stabilization factors (placental growth factor, PDGFB1, angiopoietin 1) in CC-RCC with high MVD, a group of CC-RCC highly enriched in low nuclear grade CC-RCC, with low ECP%. Our results suggest heterogeneity in angiogenic activity and vessel maturation of CC-RCC, to a large extent linked to nuclear grade, and, with probable therapeutic implications.

RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2007:449551 CAPLUS

DN 147:403661

TI Placental abnormalities in ovine somatic cell clones at term: a light and electron microscopic investigation

AU Palmieri, C.; Loi, P.; Reynolds, L. P.; Ptak, G.; Della Salda, L.

CS Department of Comparative Biomedical Sciences, Faculty of Veterinary Medicine, Teramo University, Teramo, 64100, Italy

SO Placenta (2007), 28(5-6), 577-584
CODEN: PLACDF; ISSN: 0143-4004

PB Elsevier Ltd.

DT Journal

LA English

AB To investigate the reasons for fetal losses after somatic cell nuclear transfer, an immunohistochem. and ultrastructural anal. of cloned placenta was performed. The main features obsd. were a marked redn. of villous vascularization, hypoplasia of trophoblastic epithelium, lack of binucleate cells, immaturity of placental vessels and reduced vasculogenesis. By means of transmission electron microscopy (TEM), a diffuse thickening and lamination of subtrophoblastic basement membrane (SBM) were noted in cloned placenta. These results led us to hypothesize, through an autoamplification model, that the abnormal vascularization, the ischemia and the low development of an high specialized trophoblastic epithelium were the primary causes of the fetal loss occurring after somatic cells nuclear transfer.

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 10 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2007:915719 CAPLUS
DN 148:164522
TI Different degrees f vascularization and their relationship t the expression of vascular endthelial growth factor f, placental growth factor, angiopoietins, and their receptors in first-trimester decidual tissues
AU Plaisier, Margreet; Rodrigues, Sharon; Willems, Florian; Koolwijk, Pieter; van Hinsbergh, Victor W. M.; Helmerhorst, Frans M.
CS Department of Biomedical Research, TNO-Quality of Life, Leiden, Neth.
SO Fertility and Sterility (2007), 88(1), 176-187
CODEN: FESTAS; ISSN: 0015-0282
PB Elsevier
DT Journal
LA English
AB Objective: To evaluate vascular adaptation to implantation by studying vascularization and angiogenic factors in the decidua basalis (DB), decidua parietalis, and decidual secretory endometrium of first-trimester pregnancies. Comparison of these tissues provides information about the regulation of vascularization by pregnancy-induced hormones and/or the extravillous trophoblast (EVT). Design: Prospective study. Setting: Leids University Medical Center (LUMC). Patient(s): Women (n = 32) undergoing voluntarily first-trimester termination of pregnancy. Intervention(s): Decidual samples from vacuum-aspiration. Main Outcome Measure(s): Evaluation of vascularization, detrmined by CD34 immunohistochem., and vascular endothelial growth factor-A, placental growth factor (PlGF), vascular endothelial growth factor receptor 1 (Flt-1), vascular endothelial growth factor receptor 2, angiopoietin-1 (Ang-1), angiopoietin-2 (Ang-2), and TIE-2 protein and mRNA (mRNA) expression, detd. by reverse transcriptase-polymerase chain reaction and immunohistochem., in serial paraffin sections. Results(s): Pregnancy-induced hormones and EVT influence vascularization by enhancing the vascular and luminal surface, and by reducing vessel d. at the implantation site. These changes correlate with differences in gene and protein expression. Placental growth factor mRNA and PlGF and Flt-1 protein expressions were elevated in DB under the influence of EVT. In addn., the angiopoietins were differentially expressed, in favor of Ang-2, in DB. Conclusion(s): The EVT and pregnancy-induced hormones might be assocd. with the regulation of vascularization and the expression of angiogenic factors in decidua. The induction of PlGF and Flt-1, and the Ang-2:Ang-1 ratio in DB, suggest that these factors play a role in regulating angiogenesis at the implantation site.
RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2006:504031 CAPLUS
DN 145:60613
TI Expression of Vascular Endothelial Growth Factor Receptor 1 in Bone Marrow-derived Mesenchymal Cells is Dependent on Hypoxia-inducible Factor 1
AU Okuyama, Hiroaki; Krishnamachary, Balaji; Zhou, Yi Fu; Nagasawa, Hideko; Bosch-Marce, Marta; Semenza, Gregg L.
CS Vascular Biology Program, Institute for Cell Engineering, Baltimore, MD, 21205, USA
SO Journal of Biological Chemistry (2006), 281(22), 15554-15563
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB Bone marrow-derived cells are recruited to sites of ischemia, where they promote tissue vascularization. This response is dependent upon the expression of vascular endothelial growth factor (VEGF) receptor 1 (VEGFR1), which mediates cell migration in response to VEGF or placental growth factor (PLGF). In this study, we found that exposure of cultured mouse bone marrow-derived mesenchymal stromal cells (MSC) to hypoxia or an adenovirus encoding a constitutively active form of hypoxia-inducible factor 1 (HIF-1) induced VEGFR1 mRNA and protein expression and promoted ex vivo migration in response to VEGF or PLGF. MSC in which HIF-1 activity was inhibited by a dominant neg. or RNA interference approach expressed markedly reduced levels of VEGFR1 and failed to migrate or activate AKT in response to VEGF or PLGF. Thus, loss-of-function and gain-of-function approaches demonstrated that HIF-1 activity is necessary and sufficient for basal and hypoxia-induced VEGFR1 expression in bone marrow-derived MSC.
RE.CNT 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2006:947560 CAPLUS
DN 147:91537
TI Isolation of mesenchymal stem cells from human placenta: comparison with human bone marrow mesenchymal stem cells
AU Miao, Zongning; Jin, Jun; Chen, Lei; Zhu, Jianzhong; Huang, Wei; Zhao, Jidong; Qian, Hanguang; Zhang, Xueguang
CS The Stem Cell Research Lab of Wuxi, No. 3 People's Hospital, Wuxi, 214041, Peop. Rep. China
SO Cell Biology International (2006), 30(9), 681-687

CODEN: CBIIEV; ISSN: 1065-6995
PB Elsevier B.V.
DT Journal
LA English
AB The presence within bone marrow of a population of mesenchymal stem cells (MSCs) able to differentiate into a no. of different mesenchymal tissues, including bone and cartilage, was first suggested by Friedenstein nearly 40 years ago. Since then MSCs have been demonstrated in a variety of fetal and adult tissues, including bone marrow, fetal blood and liver, cord blood, amniotic fluid and, in some circumstances, in adult peripheral blood. MSCs from all of these sources can be extensively expanded in vitro and when cultured under specific permissive conditions retain their ability to differentiate into multiple lineages including bone, cartilage, fat, muscle, nerve, glial and stromal cells. There has been great interest in these cells both because of their value as a model for studying the mol. basis of differentiation and because of their therapeutic potential for tissue repair and immune modulation. However, MSCs are a rare population in these tissues. Here we tried to identify cells with MSC-like potency in human placenta. We isolated adherent cells from trypsin-digested term placentas and examd. these cells for morphol., surface markers, and differentiation potential and found that they expressed several stem cell markers. They also showed endothelial and neurogenic differentiation potentials under appropriate conditions. We suggest that placenta-derived cells have multilineage differentiation potential similar to MSCs in terms of morphol. and cell-surface antigen expression. The placenta may prove to be a useful source of MSCs.
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 13 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2007:573690 CAPLUS
DN 147:116061
TI Relationship of angiogenesis, expression of VEGF, MMP-2 and MMP-9 in placental villi with spontaneous abortion during early pregnancy
AU Tan, Bingbing; Wang, Qianxing; Tan, Xiaoshan; Liu, Huaqing
CS Reproductive Medical Center, Affiliated Hospital, Zunyi Medical College, Zunyi, 563003, Peop. Rep. China
SO Shengzhi Yu Biyun (2006), 26(8), 477-482
CODEN: SCYYDZ; ISSN: 0253-357X
PB Shengzhi Yu Biyun Bianjibu
DT Journal
LA Chinese
AB The aim of this paper is to explore relationship between

angiogenesis, expression of VEGF, MMP-2 and -9 in human placental villi, and early spontaneous abortion. Thirty women of spontaneous abortion during early pregnancy were included in study group, 20 women of induced abortion during early pregnancy were selected as control group. Microvessel d.(MVD) in placental villi was measured by immunohistochem. method by selecting CD34 as a marker of microvessel. Expression level of VEGF mRNA in placental villi was detected by RT- PCR. Expression level of MMP-2, -9 protein in placental villi was measured by gelatin zymog. technique. Both expression of VEGF121 mRNA and MVD in spontaneous abortion group were lower than that in induced abortion group($P<0.01$) and a pos. correlation between expression of VEGF16V21 mRNA and the MVD can be showed in either spontaneous abortion group or control group(r.eta.=0.979, $P<0.001$; r.lambda.=0.918, $P<0.001$). In spontaneous abortion group the expression of pro-MMP-9 decreased obviously($P<0.0$), and the expression of active MMP-2 increased significantly, when compared with control group. The deficient angiogenesis caused by the lower expression of VEGF121 mRNA and the expressive maladjustment of MMP-2 and MMP-9 in placental villi may be concerned with spontaneous abortion during early pregnancy.

L5 ANSWER 14 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2006:643113 CAPLUS
DN 146:140432
TI Decreased expression of the angiogenic regulators CYR61 (CCN1) and NOV (CCN3) in human placenta is associated with pre-eclampsia
AU Gellhaus, Alexandra; Schmidt, Markus; Dunk, Caroline; Lye, Stephen J.; Kimmig, Rainer; Winterhager, Elke
CS Institute of Anatomy, University Hospital Essen, Essen, Germany
SO Molecular Human Reproduction (2006), 12(6), 389-399
CODEN: MHREFD; ISSN: 1360-9947
PB Oxford University Press
DT Journal
LA English
AB The pregnancy disorder pre-eclampsia (PE) is thought to be caused in part by shallow invasion of the extravillous trophoblast (EVT) leading to uteroplacental insufficiency and hypoxia. Here, we focused on the expressions of cysteine-rich 61 (CYR61, CCN1) and nephroblastoma overexpressed (NOV, CCN3), members of the CCN family of angiogenic regulators, in human placenta during normal pregnancy compared with pre-eclamptic and HELLP placenta using quant. RT-PCR, western blotting and immunocytochem. During normal pregnancy, both proteins showed increasing expression levels and were strongly coexpressed in endothelial cells of vessels, stromal cells and interstitial EVT giant cells. However, NOV showed an earlier onset of expression in villus endothelial cells during gestation compared with CYR61, which may signify

distinct roles of these proteins in placental angiogenesis. In early-onset pre-eclamptic placenta, both CYR61 and NOV were expressed at a significantly lower level compared with normal matched controls. This decrease of CYR61 and NOV in pre-eclamptic placenta is not assocd. with a decrease of the endothelial marker CD34 or vimentin. No obvious changes in the localization of CYR61 and NOV in pre-eclamptic placenta were detected but a change in the intracellular distribution in trophoblast giant cells. Our data point to a potential role of both mols. in the pathogenesis of early-onset PE.

RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 15 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2004:681685 CAPLUS

DN 141:185593

TI Methods for modulating stem cells recruitment, proliferation and differentiation with VEGF-B and PDGF and therapeutic uses thereof

IN Alitalo, Kari; Eriksson, Ulf; Carmeliet, Peter; Li, Xuri; Collen, Desire; Yla-Herttuala, Seppo; Salven, Petri; Rajantie, Iiro

PA Ludwig Institute for Cancer Research, USA; Licentia, Ltd.; Flanders Interuniversity Institute for Biotechnology

SO PCT Int. Appl., 151 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004070018	A2	20040819	WO 2004-US3316	20040204
WO 2004070018	A3	20050203		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004209668	A1	20040819	AU 2004-209668	20040204
US 20040248796	A1	20041209	US 2004-772927	20040204
EP 1594527	A2	20051116	EP 2004-708229	20040204
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006517586	T	20060727	JP 2006-503352	20040204

PRAI US 2003-445021P P 20030204
US 2003-471412P P 20030516
WO 2004-US3316 A 20040204

AB The present invention provides materials and methods for VEGF-B AND PDGF therapy, esp. therapy directed at stem cell recruitment, proliferation, and/or differentiation.

RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 16 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2004:379493 CAPLUS

DN 141:117635

TI Sequential Expression of VEGF and its Receptors in Human Placental Villi During Very Early Pregnancy: Differences Between Placental Vasculogenesis and Angiogenesis

AU Demir, R.; Kayisli, U. A.; Seval, Y.; Celik-Ozenci, C.; Korgun, E. T.; Demir-Weusten, A. Y.; Huppertz, B.

CS Faculty of Medicine, Department of Histology and Embryology, Akdeniz University, Antalya, 07070, Turk.

SO Placenta (2004), 25(6), 560-572
CODEN: PLACDF; ISSN: 0143-4004

PB Elsevier Science Ltd.

DT Journal

LA English

AB Vascularization within the human placenta is the result of the de novo formation of vessels derived from pluripotent precursor cells in the mesenchymal core of the villi. Vascularization of placental villi starts at around day 21 post conception (p.c.) with a four somite embryo. At this stage progenitors of hemangiogenic cells differentiate to form first vessels. These progenitor cells are thought to be directly derived from mesenchymal cells rather than originating from fetal blood cells. We investigated the relation between differentiation of stromal cells towards endothelial cells and vascular structures and the expression pattern of the resp. growth factors. Using transmission electron microscopy and immunohistochem. (for VEGF, Flt-1, Flk-1, CD14, CD34, and CD68) the development of placental vasculogenesis during very early stages of pregnancy (days 22-48 p.c.) was studied. We found that VEGF is strongly expressed in villous cytotrophoblast cells and subsequently in Hofbauer cells while its receptors Flt-1 and Flk-1 are found on vasculogenic and angiogenic precursor cells. The developmental expression and secretion of VEGF suggests its involvement in recruitment, maintenance and formation of first angiogenic cells and vessels. Interactions between VEGF and Flk-1 and Flt-1 may regulate placental vasculogenesis and angiogenesis in a paracrine and autocrine manner. The sequential

expression of growth factors in different cell types may point to the fact that placental vasculogenesis and angiogenesis are clearly distinct events.

RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 17 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2004:438760 CAPLUS

DN 141:137460

TI Angiogenin Distribution in Human Term Placenta, and Expression by Cultured Trophoblastic Cells

AU Pavlov, Nadine; Hatzi, Elissavet; Bassaglia, Yann; Frendo, Jean-Louis; Evain-Brion, Daniele; Badet, Josette

CS Institut National de la Sante et de la Recherche Medicale Unite, Paris, Fr.

SO Angiogenesis (2004), Volume Date 2003, 6(4), 317-330
CODEN: AGIOFT; ISSN: 0969-6970

PB Kluwer Academic Publishers

DT Journal

LA English

AB Human angiogenin is a 14-kDa secreted protein with angiogenic and ribonucleolytic activities. Angiogenin is assocd. with tumor development but is also present in normal biol. fluids and tissues. To further address the physiol. role of angiogenin, we studied its expression in situ and in vitro, using the human term placenta as a model of physiol. angiogenesis. Angiogenin was immunodetected by light and transmission electron microscopy, and its cellular distribution was established by double immunolabelling with cell markers including von Willebrand factor, platelet/endothelial cell adhesion mol.-1 (PECAM-1), CD34, Tie-2, vascular endothelial cadherin (VE-cadherin), vascular endothelial growth factor receptor-2 (VEGF-R2), erythropoietin receptor (Epo-R), alpha-smooth muscle actin, CD45, cytokeratin 7, and Ki-67. Angiogenin immunoreactivity was detected in villous and extravillous trophoblasts, the trophoblast basement membrane, the endothelial basal lamina, fetal blood vessels, fetal and maternal red blood cells, and amnionic cells. Its expression was confirmed by in situ hybridization with a digoxygenin-labeled cDNA probe and reverse transcriptase-polymerase chain reaction amplification. Villous cytotrophoblasts, isolated and differentiated in vitro into a functional syncytiotrophoblast, expressed and secreted angiogenin. Given its known biol. activities in vitro and its obsd. pattern of expression, these data suggest that, in human placenta, angiogenin has a role not only in angiogenesis but also in vascular and tissue homeostasis, maternal immune tolerance of the foetus, and host defences.

RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 18 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2003:836830 CAPLUS
DN 139:317453
TI Methods for identification of modulators of angiogenesis, compounds discovered thereby, and methods of treatment using the compounds
IN Hariri, Robert J.; Payvandi, Faribourz; Wu, Lei; Stirling, David I.; Ye, Qian
PA Celgene Corporation, USA
SO PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003086373	A1	20031023	WO 2003-US11578	20030414
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2481387	A1	20031023	CA 2003-2481387	20030414
AU 2003237078	A1	20031027	AU 2003-237078	20030414
AU 2003237078	B2	20071108		
EP 1496878	A1	20050119	EP 2003-736463	20030414
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1658848	A	20050824	CN 2003-813733	20030414
JP 2005536189	T	20051202	JP 2003-583394	20030414
NZ 536050	A	20071130	NZ 2003-536050	20030414
MX 2004009996	A	20050701	MX 2004-9996	20041012
US 20050148034	A1	20050707	US 2004-511354	20041222
PRAI US 2002-372127P	P	20020412		
WO 2003-US11578	W	20030414		

AB The invention provides methods for identifying modulators of angiogenesis using human cells. The methods of the invention can be employed to assay compds. for their ability to modulate human

angiogenesis utilizing human pluripotent stem cells in an in vitro assay system. The invention further provides methods for identifying modulators of human angiogenesis by detg. the ability of a test compd. to modulate spontaneous vasogenesis in an in vitro assay system utilizing nonembryonic pluripotent stem cells. The invention provides an in vitro assay systems using nonembryonic pluripotent stem cells for the identification of compds. that modulate human angiogenesis or human vasogenesis. The invention further provides methods of treatment which require modulation of human angiogenesis or vasogenesis, comprising administering to patients in need of such treatment compds. which have been identified to be inhibitors of human angiogenesis or vasogenesis.

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 19 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2003:133437 CAPLUS
DN 138:163608
TI Isolation and mobilization of stem cells expressing VEGFR-1
IN Rafii, Shahin; Witte, Larry
PA Imclone Systems Incorporated, USA
SO PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003014326	A2	20030220	WO 2002-US25657	20020812
WO 2003014326	A3	20030410		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2454251	A1	20030220	CA 2002-2454251	20020812
AU 2002355580	A1	20030224	AU 2002-355580	20020812
EP 1423012	A2	20040602	EP 2002-752822	20020812
EP 1423012	B1	20071114		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK
JP 2005526482 T 20050908 JP 2003-519456 20020812
AT 378056 T 20071115 AT 2002-752822 20020812
ES 2299590 T3 20080601 ES 2002-752822 20020812
US 20050026220 A1 20050203 US 2004-484511 20040708
PRAI US 2001-311705P P 20010810
WO 2002-US25657 W 20020812
AB The invention is directed to methods of isolating mammalian stem cells expressing the VEGF receptor VEGFR-1 and compns. thereof. The present invention is also directed to methods of using such isolated mammalian stem cells expressing VEGFR-1 to treat various conditions, which can involve inducing hematopoiesis, vasculogenesis and/or angiogenesis , myogenesis, and neurogenesis to treat the various condition. Finally, the present invention is directed to therapeutic methods using a mol. that binds and activates or stimulates VEGFR-1, for example, P1GF, to stimulate proliferation and/or differentiation and mobilization, i.e., motogenesis, of stem cells.

L5 ANSWER 20 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2003:42824 CAPLUS
DN 138:117632
TI Compositions and methods for inhibiting endothelial cell proliferation and regulating angiogenesis using cancer markers
IN Holaday, John W.; Fortier, Anne H.
PA USA
SO U.S. Pat. Appl. Publ., 97 pp., Cont.-in-part of U.S. Ser. No. 907,402.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030012792	A1	20030116	US 2002-131241	20020425
US 6413513	B1	20020702	US 1999-413049	19991006
US 20020137668	A1	20020926	US 2001-907402	20010717
US 6544947	B2	20030408		

PRAI US 1998-86586P P 19980522
US 1999-316802 A2 19990521
US 1999-413049 A1 19991006
US 2001-907402 A2 20010717
AB The invention provides cancer markers including prostate specific antigen (PSA), carcinoembryonic antigen (CEA), neuron specific enolase (NSE), human chorionic gonadotropin (HCG-.alpha., HCG-.beta.), cancer antigen (CA 19-9), analogs, derivs., variants, substantially homologous peptides, mimetics, agonists, antagonists, or fusion peptides of these cancer markers. In a preferred embodiment of the invention, the cancer marker is

administered with an angiogenic inhibitory peptide, a cytotoxic drug or both. Serine proteases and kallikreins exhibit potent antiangiogenic activity on human and other animal cells, particularly endothelial cells. More particularly, the use of a cancer marker, such as PSA, CEA, HCG, NSE, or CA19-9, to inhibit or ameliorate angiogenesis and angiogenesis-related diseases such as cancer, arthritis, macular degeneration, and diabetic retinopathy is disclosed.

L5 ANSWER 21 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2003:523102 CAPLUS
DN 139:274700
TI VEGF-, KIT protein- and nutral endopeptidase (NEP/CD10)-positive myofibroblasts-precursors of angiogenesis in chorioangiomas?
AU Noack, F.; Sotlar, K.; Thorns, C.; Smrcek, J.; Diedrich, K.; Feller, A. C.; Horny, H.-P.
CS Department of Pathology, University Hospital Luebeck, Luebeck, 23538, Germany
SO Placenta (2003), 24(7), 758-766
CODEN: PLACDF; ISSN: 0143-4004
PB Elsevier Science Ltd.
DT Journal
LA English
AB Chorioangiomas are benign angiomatous tumors of the placenta occurring with a frequency of approx. one per cent of all examd. placentae. Hypoxia and genetic factors are discussed to be predisposing factors for chorioangiomas. However, not much is known about the tumorigenesis of these benign tumors. Screening with various antibodies in a rare case of chorangiomatosis, we found disseminated spindle cells coexpressing vascular epithelial growth factor (VEGF), neutral endopeptidase 24.11 (NEP/CD10), and KIT protein (CD117) within the tumor stroma. A possible involvement of such factors in angiogenesis and tumorigenesis of chorioangiomas/chorangiomatosis has not been studied so far. Seven placentae with chorioangiomas (n=6) or chorangiomatosis (n=1), six normal placentae, and four cutaneous hemangiomas were analyzed immunohistochem. (ABC and APAAP methods) using antibodies against VEGF, NEP, KIT protein, as well as endothelial markers like PECAM-1 (CD31), CD34, v. Willebrand factor (factor VIII), and ulex europaeus. In addn., anal. of c-kit ' gain of function' mutation Asp 816 to Val by means of HinfI digestion and direct sequencing of semi-nested polymerase chain reaction products was performed. All chorioangiomas and hemangiomas strongly expressed the endothelial markers CD34, CD31, and FVIII, while only weak expression of ulex lectin was noted. Disseminated groups of VEGF-, NEP-, and KIT protein-pos. spindle cells, which coexpressed vimentin and smooth-muscle actin were identified as myofibroblasts in the stroma of four chorioangiomas. These spindle cells

were quantified as numerous in two and as rare in two other cases. No VEGF-pos. myofibroblasts, however, were detected in the villous stroma of normal control placentae and hemangiomas. Only scattered perivascular myofibroblasts expressing KIT protein and NEP were detected in early gestational placenta controls. In all chorioangiomas and chorangiomatosis PCR anal. failed to unveil c-kit ' gain of function' mutation Asp 816 to Val in KIT protein-pos. spindle cells. Moreover, a significant increase in mast cells was obsd. only in the hemangiomas. As expected, endothelial origin of chorioangiomas/chorangiomatosis was verified by CD31, CD34, FVIII expression. Myofibroblastic spindle cells expressing VEGF and NEP may be precursor cells in these peculiar angiomatous tumors. Although activating c-kit mutation Asp 816 to Val was not detected by PCR, the presence of KIT protein (CD117)-pos. intratumoral myofibroblastic spindle cells in chorioangiomas and chorangiomatosis might suggest involvement of the stem cell factor (SCF)-receptor in pathol. enhanced angiogenesis.

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 22 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2003:329538 CAPLUS

DN 139:34306

TI Altered tumor vessel maturation and proliferation in placenta growth factor-producing tumors: Potential relationship to post-therapy tumor angiogenesis and recurrence

AU Taylor, Alice P.; Rodriguez, Marisol; Adams, Kelly; Goldenberg, David M.; Blumenthal, Rosalyn D.

CS Garden State Cancer Center, Center for Molecular Medicine and Immunology, Belleville, NJ, USA

SO International Journal of Cancer (2003), 105(2), 158-164
CODEN: IJCNAW; ISSN: 0020-7136

PB Wiley-Liss, Inc.

DT Journal

LA English

AB Cells in human tumor xenografts express similar levels of angiogenic growth factors before treatment. After radioimmunotherapy (RAIT) surviving tumor cells upregulate angiogenic growth factors, including placenta growth factor (PIGF), in a tumor-specific pattern. To det. the role of post-treatment PIGF expression on blood vessel recovery, tumor xenografts were assayed for post-RAIT vessel d. (CD34+), proliferation (PCNA+) and maturity (SMA+ pericytes/mural cells). To further analyze the role of PIGF in blood vessel formation, PIGF-contg. Matrigel implants were also assessed in a similar manner. The xenografts producing post-treatment PIGF increased CD34+ microvessel d. 2- to 4-fold over untreated controls ($p < 0.05$) within 3 wk of RAIT

treatment. The proportion of mature microvessels (SMA+) decreased. Pericyte coverage and d. of microvessels remained stable in the tumor that expressed neither PIGF nor VEGF after treatment. Hb content of PIGF-contg. Matrigel implants was 5.7-fold that of anti-PIGF/anti-VEGF treated controls (Day 6, $p < 0.03$). The vessel d. in PIGF-implants averaged 36.8 .+-. 10.6/mm compared to 4.9 .+-. 6.5/mm2 in controls ($p < 0.001$). Vessels of PIGF-implants were lined by vWF+ cells, which were mostly flt-1+. These findings point to a role for PIGF in rapid restoration of tumor blood supply after treatment and thus, to enhanced likelihood of tumor regrowth. Likewise, the cells of primary human tumors that upregulate PIGF after treatment may be more likely to survive and form recurring tumors. Prevention of this angiogenic response to treatment may require administration of anti-angiogenic therapy during, rather than after, treatment.

RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 23 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2002:51030 CAPLUS

DN 137:45639

TI Identification of a novel membrane protein, HP59, with therapeutic potential as a target of tumor angiogenesis

AU Fu, Changlin; Bardhan, Smriti; Cetateanu, Nicolae D.; Wamil, Barbara D.; Wang, Yufen; Yan, He-Ping; Shi, Ergang; Carter, Clint; Venkov, Christo; Yakes, F. Michael; Page, David L.; Lloyd, R. Stephen; Mernaugh, Ray L.; Hellerqvist, Carl G.

CS Departments of Biochemistry, Vanderbilt University, Nashville, TN, 37232, USA

SO Clinical Cancer Research (2001), 7(12), 4182-4194
CODEN: CCREF4; ISSN: 1078-0432

PB American Association for Cancer Research

DT Journal

LA English

AB CM101, a polysaccharide isolated from the culture medium of Group B streptococcus, a neonatal pathogen, targets pathol. angiogenesis and inhibits tumor growth in mice and humans. CM101 also targets neonatal lung and adult sheep lung endothelial cells. A gene encoding a transmembrane protein that interacts with CM101 was isolated from a sheep lung endothelial cell cDNA library. The gene, termed sp55, encodes a 495-amino acid polypeptide. COS-7 cells transfected with a vector contg. sp55 express the SP55 protein-bound CM101 in a concn.-dependent manner. Stably transfected CHO cells also bound CM101. The corresponding human gene, hp59, was isolated from a human fetal lung cDNA library and had a predicted identity to SP55 of 86% over 495 amino acids. HP59 protein was shown by immunohistochem. to be present in the pathol. tumor vasculature

of the lung, breast, colon, and ovary, but not in the normal vasculature, suggesting that the protein may be crit. to pathol. angiogenesis . The hp59 gene and/or the HP59 protein was not expressed in a variety of normal tissues, but was significantly expressed in human fetal lung, consistent with the pathophysiol. of Group B streptococcus infections in neonates. Mice immunized with HP59 and SP55 peptides showed significant attenuation of tumor growth. Immunization effectively inhibited both the tumor angiogenesis and vasculogenesis processes, as evidenced by lack of both HP59- and CD34-pos. vessels. These results and the immunohistochem. data suggest a therapeutic potential for the CM101 target protein HP59 both as a drug target and as a vaccine against pathoangiogenesis.

RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT